Hepatitis B infection in multitransfused thalassaemics

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Summary

The risk of HBV infection by blood transfusion remains moderate due to the prevalence of HBV carriers in the general population of Peninsular Malaysia ranging from 3 to 6%. 23% of multitransfused thalassaemics showed HbsAg and 31% have been exposed to HBV infection. Hepatitis B vaccination should be carried out at the start of blood transfusion in all thalassaemics destined to be transfusion dependent following screening for HBV markers.

Key words: HBV infection; Chronic carriers; HBV vaccination.

Introduction

The incidence of viral hepatitis in multitransfused thalassaemic subjects is very high.[1,2] Blood transfusion has long been known to carry the risk of hepatitis because of the presence in the general population of chronic carriers of the viruses responsible for hepatitis: the viruses incriminated are hepatitis B (HBV), A, non A non B, as well as others such as cytomegalo virus (CMV), Epstein Barr virus (EBV) and Herpes virus (HV).[3,4] Methods to identify hepatitis B surface antigen (HBsAg) in the blood donors and more recently preventive treatment based on HBV vaccination has made this kind of hepatitis a less frequent posttransfusion complication in the thalassaemics. Despite the screening of blood donors for the HBsAg at the National Blood Services Centre (NBSC) in Kuala Lumpur since 1972, the risk of HBV infection by blood transfusion is present due to a moderate prevalence of HBV carrier rates ranging from 3 to 6% of the general population in Peninsular Malaysia.[5] As patients with thalassaemia major and some with haemoglobin E beta (HBeβ) thalassaemia need blood transfusions every 30 to 40 days to maintain their haemoglobin levels above 8–9 gm/dl, both these groups are at high risk of contracting HBV infection. The present study examines HBV data in a thalassaemic population in Malaysia prior to hepatitis B vaccination.

Materials and Methods

Patients: Thirty five patients with thalassaemia aged between 1 and 13 years seen at the Paediatric Department, General Hospital Kuala Lumpur, and the Haematology Clinic of the National University of Malaysia comprised the study group. All had confirmed thalassaemia (β thalassaemia = 23. Eβ thalassaemia = 12) and required blood transfusions of packed cells at intervals.

Table 1
HBV markers in 35 Multitransfused Thalassaemics

Race	Number of Cases		
	HBs Ag	Anti HBs	Anti HBc
Malays	3	1	3
Chinese	5	3	4
Total	8 (22.9)	4 (11.4)	7 (20)

*Percentages in parenthesis

Blood Donors: Patients received blood from donors at the NBSC. Donor selection included the exclusion of risk groups such as male homosexuals, drug addicts, HBsAg positive individuals and hepatitis implicated donors. From 1972 to 1978 the screening technique for HBsAg at the NBSC was by immunoelectroosmophoresis and from 1979 the more sensitive radioimmunoassay (RIA) was used.

Serology: Serum samples were coded and analysed in batches of 30 or more. Tests were performed in duplicate using commercial EIA kits (enzyme immunoabsorbant assays, Abbott Laboratories, U.S.A.) according to the manufacturer's instructions.

Results

A participant was considered to have HBV infection if he had two consecutive sera positive for HBsAg, anti Hbc, or both. Nine cases were positive for HBsAg (Tables 1 and 2). E000298, L7648/78, and L1236/80 remained positive for HBsAg for more than 6 months and were considered as HBV carriers. The earliest age showing HBsAg positively was seen in A2545/86, a four year old Malay male with HBEβ thalassaemia and a blood transfusion record of 11 units (prior to receiving the first blood transfusion he was HBsAg negative). The mothers of Malay children positive for HBsAg showed no HBV markers. In contrast the mothers of 3 Chinese patients showed HBV markers (HBsAg = 2; Anti HBs = 1).

Table 2
HBV markers and units of blood transfused in 35 multitransfused thalassaemics

No.	Code	Thalassaemia	Age	Sex	Race	HBs Ag	Anti Hbs	Anti Hbc	Units Blood
1	T2040	EB	1	F	M	–	–	+	2
2	L2364/87	B	2	M	C	–	–	–	3
3	M3204/85	B	2	M	M	–	–	–	5.3
4	N3527/87	B	2	F	M	–	–	–	1.1
5	N1601/87	EB	2	F	M	–	–	–	3
6	A1808/85	B	3	F	C	–	–	–	2
7	C3179/86	B	3	F	C	–	–	–	1.5
8	M1250/86	B	3	M	M	–	–	–	4.2
9	N2053/86	EB	3	F	M	–	–	–	2
10	S2391/86	EB	3	F	M	–	–	–	4
11	A2545/86	EB	4	M	M	+	–	–	11
12	L2981/84	B	4	F	C	–	–	–	10
13	K4807/86	EB	5	M	M	+	–	–	3
14	I945/87	B	5	F	M	–	–	–	17.2
15	S463/85	EB	5	M	M	–	–	–	14.0
16	H2740/80	B	6	F	C	–	–	–	26.1
17	S1614/85	EB	6	F	M	+	–	+	11.0
18	W2191/84	B	6	M	C	–	–	–	23.1
19	C7305/81	B	7	F	C	–	–	–	31
20	L5390/86	EB	7	M	C	–	+	+	24
21	L157/81	B	7	F	C	+	–	–	33.2
22	U3205/85	B	7	F	M	–	+	+	38.1
23	L123/82	B	8	F	C	–	–	–	42
24	S1837/80	B	8	M	M	–	–	–	48.5
25	E000298	EB	8	F	C	+	–	–	52.0
26	A1158/84	B	9	F	M	–	–	–	34
27	A316/83	B	10	F	M	–	–	–	64
28	C16/82	B	10	M	C	–	–	–	61
29	H2603/80	B	10	M	C	–	–	–	43.8
30	L7648/78	B	10	M	C	+	–	+	65.0
31	L5642/80	B	10	M	C	+	–	+	70.0
32	S3058/82	B	10	M	C	–	+	–	46.5
33	L1236/80	B	11	M	C	+	+	+	56.8
34	S1836/80	EB	11	M	M	–	–	–	45.0
35	M158406	EB	13	F	M	–	–	–	83

B = β Thalassaemia *EB = Eβ Thalassaemia* *M = Malays* *C = Chinese*

Discussion

In the multitransfused thalassaemics liver damage can be attributed to both viral hepatitis and siderosis[6] and intensive chelation therapy will reduce organ damage due to iron overload.[7] The high price of deferrioxamine (Desferal) the chelating agent currently used in transfusion dependent thalassaemics makes it unavailable to many. Iron loading increases with age and the possibility of contact with HBV infection and siderosis are inseparable factors in causing liver damage : siderosis facilitating the development of chronic hepatitis in the thalassaemics exposed to viruses.[8] There is a strong association of HBV infection and primary hepatocellular cancer (PHC).[9] In a nine year follow up of transfusion dependent thalassaemics there has been no case of PHC. Chronic exposure to HBV is limited by the survival period of the thalassaemics who are transfusion dependent. In the absence of chelation therapy, death occurs in the majority by the age of 15 years as a consequence of extensive organ damage secondary to siderosis.

Studies in Malaysia have shown a moderate prevalence of HBV infection ranging from 3 to 11% depending on the ethnic group of Peninsular Malaysia.[5] To ensure safe blood transfusion, screening for HbsAg was introduced in the NBSC in Kuala Lumpur from 1972. Inspite of careful HBsAg screening which is now carried out on all blood donors, in our study 23% of the multitransfused thalassaemics were HBsAg positive and 31% have been exposed to HBV infection. In some cases the HBsAg positive donors may not be identified at screening. On the other hand the thalassaemics might get their HBV infection by routes other than transfusion. Transmission from mother to neonate during birth is one of the most efficient mode of HBV transmission.[10] In our study 3 mothers showed evidence of HBV infection, however their HBV status at the time of delivery was not available.

Screening for HBV markers is vital at the start of blood transfusion treatment in all thalassaemics who are destined to be transfusion dependent and hepatitis B vaccination should be carried out in susceptible children.

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